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The prognostic value of the triglyceride-glucose index in predicting recurrence of acute pancreatitis: a retrospective cohort study

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Abstract

Background Investigating the risk of acute pancreatitis (AP) recurrence is crucial because it affects public health and medical resources. The triglyceride–glucose index (TyG-i) is recognized as a reliable marker of insulin resistance (IR), which occurs after an AP attack. However, the predictive value of the TyG-i during the first AP for subsequent recurrence remains unclear.

Methods Patients with their first AP episode between January 2014 and December 2023 were followed up retrospectively. Data on demographic characteristics, imaging findings, and laboratory examinations of their first episode and recurrences were collected. The TyG-i was calculated as follows: ln [fasting triglyceride (mg/dL) × fasting glucose (mg/dL)/2]. Factors associated with AP were evaluated using Cox regression analyses.

Results A total of 853 patients were enrolled in our study, 180 (21.1%) of whom experienced a recurrence after the first AP episode. The recurrence rate was higher in the high TyG-i index group ($n = 111$, 26.0%) than in the low TyG-i index group ($n = 69$, 16.2%; $P < 0.001$). Cox regression analyses revealed TyG-i as an independent predictor of AP recurrence in all etiologies (hazard ratio [HR] = 1.535, $P = 0.007$), as well as for the recurrence of acute biliary pancreatitis (HR = 1.829, $P = 0.035$).

Conclusion TyG-i status at the first AP episode could independently predict recurrence.

Keywords Acute pancreatitis, Triglyceride-glucose index, Insulin resistance, Recurrence, Predict

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Introduction

Acute pancreatitis (AP) is an inflammatory disease caused by abnormal activation of pancreatic enzymes, leading to damage to the pancreas, surrounding tissues, and other organs. Its incidence is rising globally [1]. Most patients with AP have clear diagnoses, mild conditions, and favorable prognoses. However, 18.6% of patients experienced AP recurrence after achieving complete or near-complete recovery [2]. Recurrent acute pancreatitis (RAP) generally requires hospitalization, resulting in increased medical costs and a greatly reduced the quality of life. In addition, almost 8% of patients with RAP developed chronic pancreatitis, in which pancreatic tissue and function were irreversible damaged [3]. Therefore, identifying the risk factors for RAP is essential for maintaining health and reducing the burdens on the healthcare systems.

Insulin resistance (IR) refers to a decline in insulin sensitivity that does not effectively promote glucose uptake and utilization. In recent years, IR has been identified as a marker of systemic inflammation. Elevated glucose and (or) lipid levels in the blood trigger the release of various pro-inflammatory mediators, such as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 [4, 5]. These mediators stimulate IR by interfering with the insulin signaling pathway [4, 5].

The triglyceride–glucose index (TyG-i), a combination of fasting blood glucose and triglyceride levels, has been proposed as a reliable indirect marker of IR [6, 7, 8]. Its use is convenient, fast and affordable, and the TyG-i has been proven to be an effective predictor of adverse cardiovascular outcomes [9, 10, 11]. However, although the TyG-i can predict severe AP [12], its value in predicting AP recurrence remains unclear. Therefore, we aimed to investigate its value in predicting RAP.

Methods

Study design and participants

This retrospective cohort study assessed the predictive value of TyG-i for recurrence after the first AP episode in patients at the First Affiliated Hospital of Sun Yat-sen University between January 2014 and December 2023. The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Sun Yat-sen University (No. [2024]495). This study was performed following the Declarations of Helsinki. The inclusion criterion was a first episode of AP which was meeting the diagnostic criteria for AP. The exclusion criteria included: < 18 years old, during pregnant, suffered from pancreatic tumors or other malignant tumors, incomplete clinical examination records, and RAP admission records. The informed consent was waived by the ethics committee.

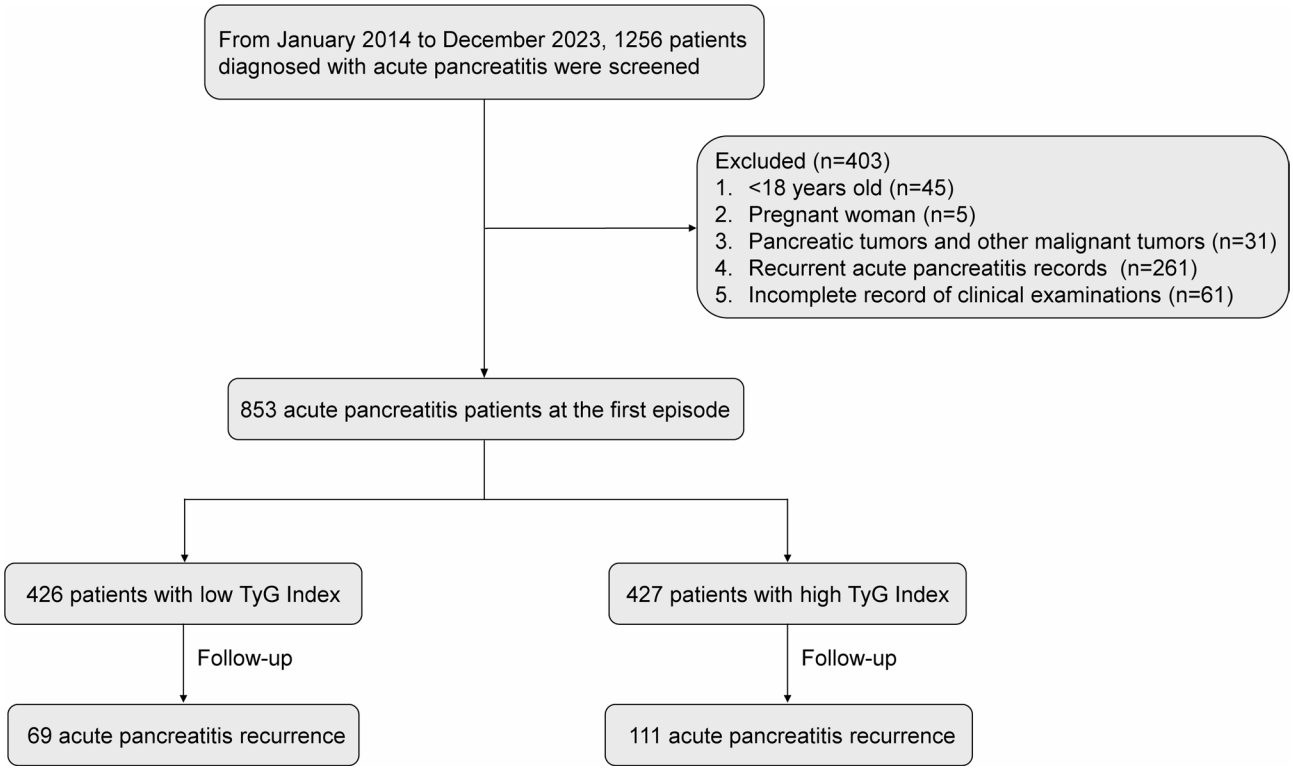

Fig. 1 Flow chart of this study. TyG-i, triglyceride-glucose index

Definitions

AP was diagnosed when at least two of the following criteria were met according to the Atlanta criteria: typical abdominal pain, serum amylase or lipase level > three times of the upper limit of normal value, and typical imaging findings of AP on abdominal ultrasonography/computed tomography (CT) [13]. The definition of RAP required the following: complete or nearly complete recovery from the first episode of AP, internal recurrence greater than 3 months, and absence of chronic pancreatitis [14]. Acute biliary pancreatitis was defined as AP induced by cholelithiasis or choledocholithiasis combined with abnormal liver function [15]. Hypertriglyceridemic acute pancreatitis was defined as AP with a serum triglyceride level greater than 11.3 mmol/L or 1000 mg/dL on admission, without bile stones or obstruction, alcohol use, or other reasons [16]. Fatty liver is noninvasively assessed by ultrasound imaging, which is the most commonly used imaging technique for diagnosing fatty liver [17]. TyG-i was calculated using the formula TyG-i = ln [fasting triglyceride (mg/dL) × fasting glucose (mg/dL)/2]. The fasting blood glucose and triglyceride levels in the formula were obtained after ≥ 8 h of fasting within 24 h of admission. Systemic inflammatory response syndrome (SIRS) was diagnosed when at least two of the following criteria were met: temperature > 38 °C or < 36 °C, heart rate > 90 beats/min, respiratory rate > 20/min or PaCO2 < 32 mmHg, and peripheral blood white blood cell (WBC) count > 12 × 10^9/L or < 4 × 10^9/L [18].

Data collection

Data on baseline characteristics were collected from medical records, including demographic features (age, gender, smoking history, and drinking history), body mass index (BMI), length of hospital stay, intensive care unit (ICU) admission, and comorbidities (diabetes, hypertension, stroke, coronary heart disease, fatty liver, and chronic obstructive pulmonary disease).

The modified computed tomography severity index (MCTSI), which reflects organ failure and extrapancreatic complications, was assessed using CT scan. According to the Atlanta classification, AP is divided into mild acute pancreatitis (MAP), moderate severe acute pancreatitis (MSAP), and severe acute pancreatitis (SAP) [13].

Local complications (acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection [ANC], walled-off necrosis, and infected pancreatic necrosis) were analyzed using CT, magnetic resonance imaging, or ultrasonography.

Systemic complications included ascites/peritonitis, portal hypertension, intra-abdominal infection, acute respiratory distress syndrome, acute kidney injury, SIRS, pulmonary/urinary infection, gastrointestinal bleeding, multiple organ dysfunction syndrome, septicemia, abdominal compartment syndrome, pancreatic encephalopathy, intestinal obstruction, and shock.

Laboratory examinations (routine blood test, serum liver enzymes, liver metabolic function, liver and kidney function, serum lipids, activated partial thrombin time [APTT], fibrinogen [Fg]), and clinical treatment strategies (low-molecular-weight heparin (LMWH) treatment, continuous renal replacement therapy [CRRT], and antibiotic treatment) were collected.

Recording of follow-up data

Follow-up was conducted by reviewing medical records until September 30, 2024. Data on the basic conditions (age, length of hospital stay, and ICU admission), severity (MCTSI score and the Atlanta classification), local and systemic complications, and laboratory examinations (routine blood test, APTT, and Fg) of patients at the first AP recurrence were collected.

Statistical analysis

Data analysis was performed by SPSS software (version 25.0; Chicago, IL, USA). A two-sided P value < 0.05 was considered statistically significant. Continuous variables are displayed as means ± standard deviations or medians and quartile ranges, whereas categorical variables are displayed as numbers and percentages. The comparison of continuous variables used the Student's t test or Mann-Whitney U test. The comparison of categorical variables used the chi-square test, continuity correction, or Fisher's exact test, as appropriate. The cumulative risk of AP recurrence after the first episode was calculated using the Kaplan-Meier method. Variables with a univariate P value < 0.05 were included in the forward LR-based multivariate Cox proportional hazards regression analysis.

Results

A total of 1256 candidates were screened, and finally 853 patients were enrolled in our study (Fig. 1). The median follow-up time was 52.2 months. The median age of the entire cohort was 45 (35–57) years old; and 528 (61.9%) cases were male. The recurrence rate after the first AP episode was 21.1%. The median TyG-i value was 9.02 (8.42–9.95).

General characteristics of the first AP episode

The participants were divided into two groups depending on the median TyG-i value (9.02) of the cohort as follows: low TyG-i group (TyG-i < 9.02; $n = 426$) and high TyG-i group (TyG-i ≥ 9.02; $n = 427$). Table 1 presents the general characteristics of the two groups. The median TyG-i values of the low and high TyG-i groups were 8.4 (8.1–8.7) and 9.9 (9.4–10.8), respectively. Compared with the low TyG-i group, the high TyG-i group exhibited younger age, a higher proportion of male patients, and higher rates

Table 1 General characteristics of the first episode of AP

Variables	Overall (*n* = 853)	Low TyG-i (*n* = 426)	High TyG-i (*n* = 427)	*P* value
Demographic variables				
Age (years)	45 (35–57)	49 (36–59)	42 (34–53)	< 0.001
Male (n, %)	528 (61.9)	243 (57.0)	285 (66.7)	0.004
Smoking history (n, %)	238 (27.9)	86 (20.2)	152 (35.6)	< 0.001
Drinking history (n, %)	206 (24.2)	73 (17.1)	133 (31.1)	< 0.001
BMI (kg/m^2)	23.9 ± 4.3	22.2 ± 3.6	25.7 ± 4.3	0.072
Comorbidity				
Diabetes (n, %)	172 (20.2)	20 (4.7)	152 (35.6)	< 0.001
Hypertension (n, %)	170 (19.9)	79 (18.5)	91 (21.3)	0.312
Stroke (n, %)	7 (0.8)	5 (1.2)	2 (0.5)	0.287
Fatty liver (n, %)	353 (41.4)	88 (20.7)	265 (62.1)	< 0.001
Coronary heart disease (n, %)	17 (2.0)	5 (1.2)	12 (2.8)	0.087
COPD (n, %)	8 (0.9)	6 (1.4)	2 (0.5)	0.177
Laboratory index				
WBC (×10^9/L)	10.7 (7.3–14.3)	9.5 (6.4–12.4)	12.0 (9.0-15.7)	< 0.001
Neutrophil (×10^9/L)	8.6 (5.0–12.0)	7.2 (4.1–10.4)	9.9 (6.7–13.2)	< 0.001
Lymphocyte (×10^9/L)	1.3 (0.9–1.8)	1.3 (0.9–1.8)	1.3 (1.0-1.8)	< 0.001
Monocyte (×10^9/L)	0.6 (0.4–0.8)	0.5 (0.4–0.7)	0.6 (0.4–0.9)	< 0.001
HB (g/L)	137 (121–151)	132 (119–145)	143 (124–158)	< 0.001
RDWCV, %	13.0 (13.0–14.0)	13.0 (13.0–14.0)	13.0 (13.0–14.0)	0.558
PLT (×10^9/L)	233 (193–287)	231.0 (191–285)	236 (195–289)	0.515
NLR	6.1 (3.3–11.1)	5.1 (2.7–10.0)	7.2 (4.0-11.8)	< 0.001
Urea (mmol/L)	4.6 (3.5-6.0)	4.6 (3.4-6.0)	4.6 (3.6-6.0)	0.822
Creatinine (μmol/L)	65 (53–78)	65 (54–78)	64 (53–79)	0.819
Uric acid (μmol/L)	337 (245–428)	332 (236–422)	344 (247–439)	0.350
Fasting blood glucose (mmol/L)	6.6 (5.4–8.8)	5.7 (4.9–6.8)	8.2 (6.4–11.7)	< 0.001
Calcium (mmol/L)	2.20 (2.10–2.30)	2.23 (2.13–2.30)	2.20 (2.10–2.30)	< 0.001
ALT (U/L)	34 (21–67)	32 (20–95)	35 (23–57)	0.829
AST (U/L)	31 (23–55)	30 (22–63)	32 (24–51)	0.463
TC (mmol/L)	4.6 (3.7–5.8)	4.1 (3.4–4.9)	5.4 (4.3–7.2)	< 0.001
TG (mmol/L)	1.6 (0.9–3.4)	1.0 (0.7–1.2)	3.4 (2.1-7.0)	< 0.001
HDL-c (mmol/L)	0.9 (0.7–1.2)	1.0 (0.8–1.3)	0.9 (0.6–1.1)	< 0.001
LDL-c (mmol/L)	2.9 (2.3–3.6)	2.6 (2.1–3.1)	3.2 (2.5-4.0)	< 0.001
APTT (s)	29.7 (26.7–34.2)	29.3 (26.4–33.4)	30.5 (27.0-35.5)	0.010
Fg (g/L)	4.0 (3.0-5.2)	3.5 (2.7–4.7)	4.6 (3.5–6.3)	< 0.001
TyG-i	9.0 (8.4–10.0)	8.4 (8.1–8.7)	9.9 (9.4–10.8)	< 0.001
Clinical course				
Median follow-up (months)	52.2 (47.0-57.4)	51.7 (44.9–58.5)	53.0 (44.3–61.8)	0.832
Recurrence (n, %)	180 (21.1)	69 (16.2)	111 (26.0)	< 0.001
Recurrence times	3 (2–4)	3 (2–4)	2 (2–4)	0.731
Hospitalization length (Days)	7 (4–10)	6 (4–10)	7 (5–11)	0.002
ICU (n, %)	48 (5.6)	14 (3.3)	34 (8.0)	0.003
Severity				
MCTSI				< 0.001
Mild (n, %)	437 (51.2)	245 (57.5)	192 (45.0)	
Moderate (n, %)	353 (41.4)	148 (34.7)	205 (48.0)	
Severe (n, %)	36 (4.2)	17 (4.0)	19 (4.4)	
Not assessable (n, %)	27 (3.2)	16 (3.8)	11 (2.6)	
Severity of AP				0.235
MAP (n, %)	748 (87.7)	381 (89.4)	367 (85.9)	
MSAP (n, %)	93 (10.9)	41 (9.6)	52 (12.2)	
SAP (n, %)	12 (1.4)	4 (0.9)	8 (1.9)	

Table 1 (continued)

Variables	Overall (*n* = 853)	Low TyG-i (*n* = 426)	High TyG-i (*n* = 427)	*P* value
Etiology				
acute biliary pancreatitis (n, %)	306 (35.9)	180 (42.2)	126 (29.5)	< 0.001
HTG AP (n, %)	64 (7.5)	0 (0.0)	64 (15.0)	< 0.001
Local complication				
Acute peripancreatic fluid collection (n, %)	108 (12.7)	45 (10.6)	63 (14.8)	0.066
Pancreatic pseudocyst (n, %)	74 (8.7)	43 (10.1)	31 (7.3)	0.141
ANC (n, %)	31 (3.6)	13 (3.1)	18 (4.2)	0.364
Walled-off necrosis (n, %)	46 (5.4)	20 (4.7)	26 (6.1)	0.367
Infected pancreatic necrosis (n, %)	2 (0.2)	0 (0.0)	2 (0.5)	0.499
Systemic complication				
Ascites/peritonitis (n, %)	233 (27.3)	104 (24.4)	129 (30.2)	0.057
Portal hypertension (n, %)	16 (1.9)	5 (1.2)	11 (2.6)	0.131
Intra-abdominal infection (n, %)	4 (0.5)	0 (0.0)	4 (0.9)	0.124
ARDS (n, %)	19 (2.2)	7 (1.6)	12 (2.8)	0.248
AKI (n, %)	6 (0.7)	3 (0.7)	3 (0.7)	1.000
SIRS (n, %)	96 (11.3)	30 (7.0)	66 (15.5)	< 0.001
Pulmonary/urinary infection (n, %)	59 (6.9)	20 (4.7)	39 (9.1)	0.011
Gastrointestinal bleeding (n, %)	6 (0.7)	2 (0.5)	4 (0.9)	0.686
Other complications (n, %)	20 (2.3)	6 (1.4)	14 (3.3)	0.071
Treatment				
Low molecular weight heparin (n, %)	141 (16.5)	42 (9.9)	99 (23.2)	< 0.001
CRRT (n, %)	23 (2.7)	3 (0.7)	20 (4.7)	< 0.001
Antibiotic therapy (n, %)	635 (74.4)	302 (70.9)	333 (78.0)	0.018
Drainage or necrosectomy (n, %)	58 (6.8)	28 (6.6)	30 (7.0)	0.793

Note: Values are presented as means ± standard deviation, median (interquartile range) or n (%). Mild (MCTSI score 0 and 2), moderate (MCTSI score 4 and 6), severe (MCTSI score 8 and 10). Other complications refer to MODS, septicemia, abdominal compartment syndrome, pancreatic encephalopathy, intestinal obstruction, shock

Abbreviations: AP, acute pancreatitis; TyG-i, triglyceride-glucose index; BMI; body mass index; COPD, chronic obstructive pulmonary disease; WBC, white blood cell; HB, hemoglobin; RDWCV, red blood cell distribution width coefficient of variability; PLT, platelet; NLR, the ratio of neutrophil to lymphocyte; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TC, total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; APTT, activated partial thromboplastin time; Fg, fibrinogen; ICU; intensive care unit; MCTSI; modified CT severity index; MAP, mild acute pancreatitis; MSAP, moderately severe acute pancreatitis; SAP, severe acute pancreatitis; HTG AP, hypertriglyceridemic acute pancreatitis; ANC, acute necrotic collection; ARDS, acute respiratory distress syndrome; AKI, acute kidney injury; SIRS, systemic inflammatory response syndrome; CRRT, continuous renal replacement therapy; MODS, multiple organ dysfunction syndrome

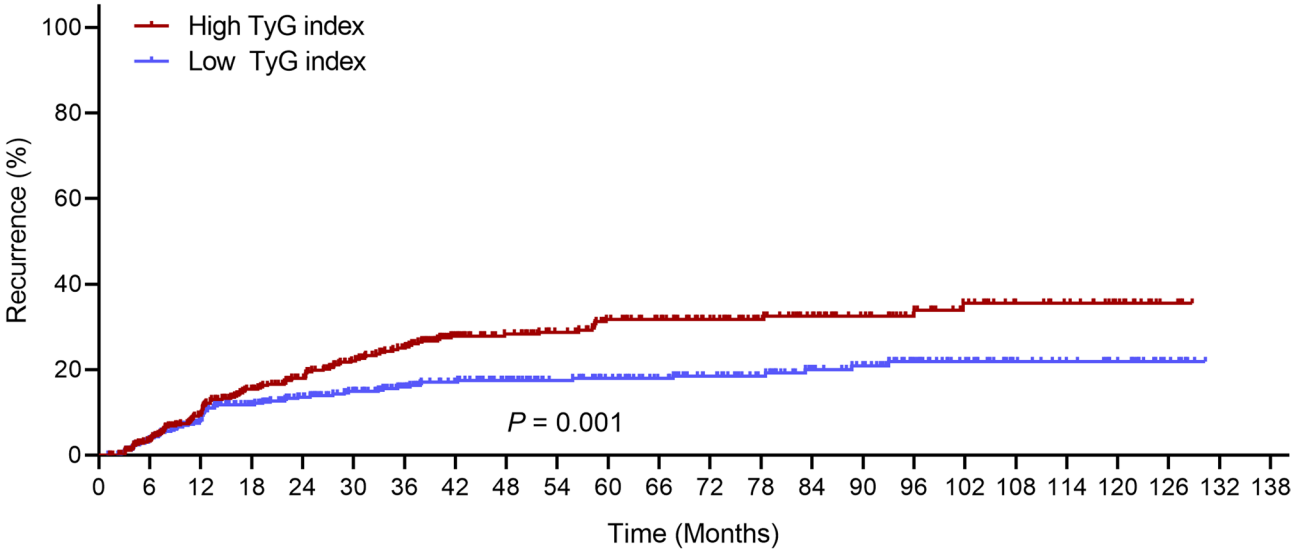

Fig. 2 Cumulative risk of the recurrence after the first episode of acute pancreatitis of all etiologies based on the TyG-i. TyG-i, triglyceride-glucose index

Table 2 Cox regression analysis of prognostic factors for RAP of all etiologies

Variables	Univariate		Multivariate	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Age (≥ 45/<45 years)	0.658 (0.489–0.885)	0.006		
Gender (Male/female)	1.863 (1.332–2.604)	< 0.001	1.780 (1.247–2.542)	0.002
Smoking history (Positive/Negative)	1.837 (0.363–2.476)	0.579		
Drinking history (Positive/Negative)	1.128 (0.808–1.572)	0.479		
BMI (≥ 23.9/<23.9 kg/m^2)	1.380 (0.814–2.338)	0.231		
Diabetes (Present/Absent)	1.500 (1.075–2.092)	0.017		
Hypertension (Present/Absent)	1.066 (0.748–1.520)	0.723		
Stroke (Present/Absent)	0.567 (0.079–4.046)	0.571		
Fatty liver (Present/Absent)	1.565 (1.168–2.097)	0.003		
Coronary heart disease (Present/Absent)	0.542 (0.134–2.182)	0.388		
COPD (Present/Absent)	0.049 (0.000-18.361)	0.318		
WBC (≥ 10.7/<10.7 × 10^9/L)	0.970 (0.723–1.303)	0.842		
Neutrophil (≥ 8.6/<8.6 × 10^9/L)	0.979 (0.729–1.314)	0.888		
Lymphocyte (≥ 1.3/<1.3 × 10^9/L)	1.356 (1.005–1.828)	0.046		
Monocyte (≥ 0.6/<0.6 × 10^9/L)	0.951 (0.706–1.280)	0.739		
HB (≥ 137/<137 g/L)	1.677 (1.236–2.274)	< 0.001		
RDWCV (≥ 13/<13%)	1.133 (0.782–1.640)	0.509		
PLT (≥ 233/<233 × 10^9/L)	1.033 (0.768–1.388)	0.832		
NLR (≥ 6.1/<6.1)	0.870 (0.648–1.169)	0.356		
Urea (≥ 4.6/<4.6 mmol/L)	1.009 (0.752–1.354)	0.950		
Creatinine (≥ 65/<65 μmol/L)	1.170 (0.871–1.571)	0.296		
Uric acid (≥ 337/<337 μmol/L)	0.886 (0.539–1.457)	0.634		
Calcium (≥ 2.2/<2.2 mmol/L)	1.277 (0.929–1.754)	0.131		
ALT (≥ 34/<34 U/L)	0.818 (0.609–1.098)	0.181		
AST (≥ 31/<31 U/L)	0.752 (0.560–1.010)	0.058		
APTT (≥ 29.7/<29.7 s)	1.673 (1.232–2.272)	< 0.001	1.558 (1.141–2.128)	0.005
Fg (≥ 4/<4 g/L)	0.815 (0.601–1.105)	0.188		
TyG-i (≥ 9.02/<9.02)	1.645 (1.218–2.222)	0.001	1.535 (1.122–2.098)	0.007
Hospital stay (≥ 5/<5 days)	0.809 (0.589–1.111)	0.191		
ICU (Admission/Not)	1.075 (0.584–1.978)	0.816		
MCTSI (Severe/Moderate and mild)	0.434 (0.139–1.359)	0.152		
Severity of AP (Severe/Moderate and mild)	1.143 (0.365–3.579)	0.818		
Acute biliary pancreatitis (Positive/Negative)	0.838 (0.605–1.161)	0.288		
Acute peripancreatic fluid collection (Present/Absent)	0.901 (0.566–1.434)	0.660		
Pancreatic pseudocyst (Present/Absent)	1.313 (0.807–2.137)	0.273		
ANC (Present/Absent)	2.448 (1.362–4.399)	0.003	1.962 (1.062–3.624)	0.031
Walled-off necrosis (Present/Absent)	1.091 (0.558–2.133)	0.799		
Infected pancreatic necrosis (Present/Absent)	0.049 (0.000-2763.195)	0.590		
Ascites/peritonitis (Present/Absent)	0.826 (0.587–1.164)	0.276		
Portal hypertension (Present/Absent)	1.978 (0.812–4.815)	0.133		
Intra-abdominal infection (Present/Absent)	2.355 (0.584–9.495)	0.229		
ARDS (Present/Absent)	0.460 (0.114–1.853)	0.275		
AKI (Present/Absent)	0.965 (0.135–6.890)	0.971		
SIRS (Present/Absent)	1.030 (0.653–1.624)	0.900		
Pulmonary/urinary infection (Present/Absent)	0.595 (0.293–1.210)	0.152		
Gastrointestinal bleeding (Present/Absent)	0.962 (0.135–6.870)	0.969		
Other complications (Present/Absent)	0.786 (0.251–2.462)	0.680		
Low molecular weight heparin (Use/Not)	0.810 (0.531–1.236)	0.328		
CRRT (Use/Not)	1.062 (0.436–2.583)	0.895		

Table 2 (continued)

Variables	Univariate		Multivariate	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Antibiotic therapy (Use/Not)	0.877 (0.634–1.214)	0.430		
Drainage or necrosectomy (Use/Not)	0.982 (0.519–1.860)	0.956		

Note: Other complications refer to MODS, septicemia, abdominal compartment syndrome, pancreatic encephalopathy, intestinal obstruction, shock

Abbreviations: RAP, recurrent acute pancreatitis; HR, hazard ratio; CI, confidence interval; BMI; body mass index; COPD, chronic obstructive pulmonary disease; WBC, white blood cell; HB, hemoglobin; RDWCV, red blood cell distribution width coefficient of variability; PLT, platelet; NLR, the ratio of neutrophil to lymphocyte; ALT, alanine aminotransferase; AST, aspartate aminotransferase; APTT, activated partial thromboplastin time; Fg, fibrinogen; TyG-i, triglyceride-glucose index; ICU; intensive care unit; MCTSI; modified CT severity index; AP, acute pancreatitis; ANC, acute necrotic collection; ARDS, acute respiratory distress syndrome; AKI, acute kidney injury; SIRS, systemic inflammatory response syndrome; CRRT, continuous renal replacement therapy; MODS, multiple organ dysfunction syndrome

of smoking and drinking (all $P<0.050$). The high TyG-i group had a higher prevalence of diabetes (35.6%) and fatty liver (62.1%) than the low TyG-i group (all $P<0.001$).

The high TyG-i group exhibited higher WBC, neutrophil, lymphocyte, and monocyte counts, and higher levels of hemoglobin (HB), neutrophil to lymphocyte ratio (NLR), APTT, and Fg (all $P<0.050$). The high TyG-i group had longer hospital stays, more ICU admissions, and more severe the MCTSI (all $P<0.050$). Moreover, acute biliary pancreatitis was noted in 42.4% of the low TyG-i group, and only 29.5% of the high TyG-i group ($P<0.001$). No statistically significant difference in local complications was observed between the two groups; however, the incidence of SIRS (15.5%) and pulmonary/urinary infection (9.1%) were higher in the high TyG-i group than in the low TyG-i group (all $P<0.050$).

In terms of treatment, the high TyG-i group received more LMWH (23.2%), CRRT treatment (4.7%), and antibiotics treatments (78.0%) than the low TyG-i group. The high TyG-i group used more third-generation cephalosporins (59.0%), carbapenems (18.3%), penicillins (12.4%), and antifungal agents (5.9%) (all $P<0.050$) than the low TyG-i group (Supplementary Table 1). No statistically significant difference in the drainage or necrosectomy was observed between the two groups.

Predictive value of TyG-i for RAP of all etiologies

The median follow-up times of the low and high TyG-i groups were 51.7 months and 53.0 months, respectively ($P=0.832$; Table 1). The recurrence rate was higher in the high TyG-i group ($n=111$, 26.0%) than in the low TyG-i group ($n=69$, 16.2%; $P<0.001$; Table 1).

As is shown in Fig. 2, the high TyG-i group did not reach a recurrence rate of 50%, and the recurrence rates reached 25% at 36 months. The low TyG-i group did not reach either a 50% or 25% recurrence rate. Univariate Cox regression analyses showed that the TyG-i (hazard ratio [HR] = 1.645, 95% confidence interval [CI] 1.218–2.222, $P=0.001$) was a significant risk factor for RAP, and multivariate Cox regression analyses demonstrated that the TyG-i (HR = 1.535, 95% CI 1.122–2.098, $P=0.007$) was an independent predictor of RAP (Table 2).

Other predictive factors for RAP of all etiologies

In addition to TyG-i, univariate Cox regression analyses revealed other risk factors for RAP, including male sex (HR = 1.863, 95% CI 1.332–2.604, $P<0.001$), APTT ≥ 29.7s (HR = 1.673, 95% CI 1.232–2.272, $P<0.001$), and ANC (HR = 2.448, 95% CI 1.362–4.399, $P=0.003$) (Table 2). Multivariate Cox regression analyses revealed that male sex (HR = 1.780, 95% CI 1.247–2.542, $P=0.002$), APTT ≥ 29.7s (HR = 1.558, 95% CI 1.141–2.128, $P=0.005$), and ANC (HR = 1.962, 95% CI 1.062–3.624, $P=0.031$) were independent predictors of RAP (Table 2).

Survival analysis indicated that the recurrence rate in males was higher than that in females in our cohort, although neither group had a recurrence rate of 50% ($P<0.001$; Fig. 3A). The recurrence rate in males reached 25% at 36.1 months, whereas that in females did not reach 25%. Similarly, the recurrence rate in the ANC presence group was higher than that in the absence group, with neither group reaching a recurrence rate of 50% ($P=0.002$; Fig. 3B). The time to a 25% recurrence was 18.4 months for the ANC presence group compared to 80.3 months for the absence group. Moreover, the recurrence rate of the APTT ≥ 29.7s group was higher than that in the APTT < 29.7s group, and neither group reached a recurrence rate of 50% ($P<0.001$; Fig. 3C). The recurrence rate in the APTT ≥ 29.7s group reached 25% at 35.7 months, whereas the APTT < 29.7s group did not reach a recurrence rate of 25%.

Predictive value of TyG-i for the recurrence of acute biliary pancreatitis

As biliary etiology predominates in AP, therefore, we further analyzed the value of the TyG-i in the recurrence of acute biliary pancreatitis. The general characteristics of the first episode of acute biliary pancreatitis are listed in the Supplementary Table 2. The recurrence rate of acute biliary pancreatitis was higher in the high TyG-i group ($n=29$, 23.0%) than that in the low TyG-i group ($n=22$, 12.2%; $P=0.013$). As is shown in Fig. 4, the high TyG-i group did not reach a recurrence rate of 50%, although the recurrence rate reached 25% at 58.4 months. The low TyG-i group did not reach a recurrence rate of 50% or 25%. Univariate Cox regression analyses showed that the

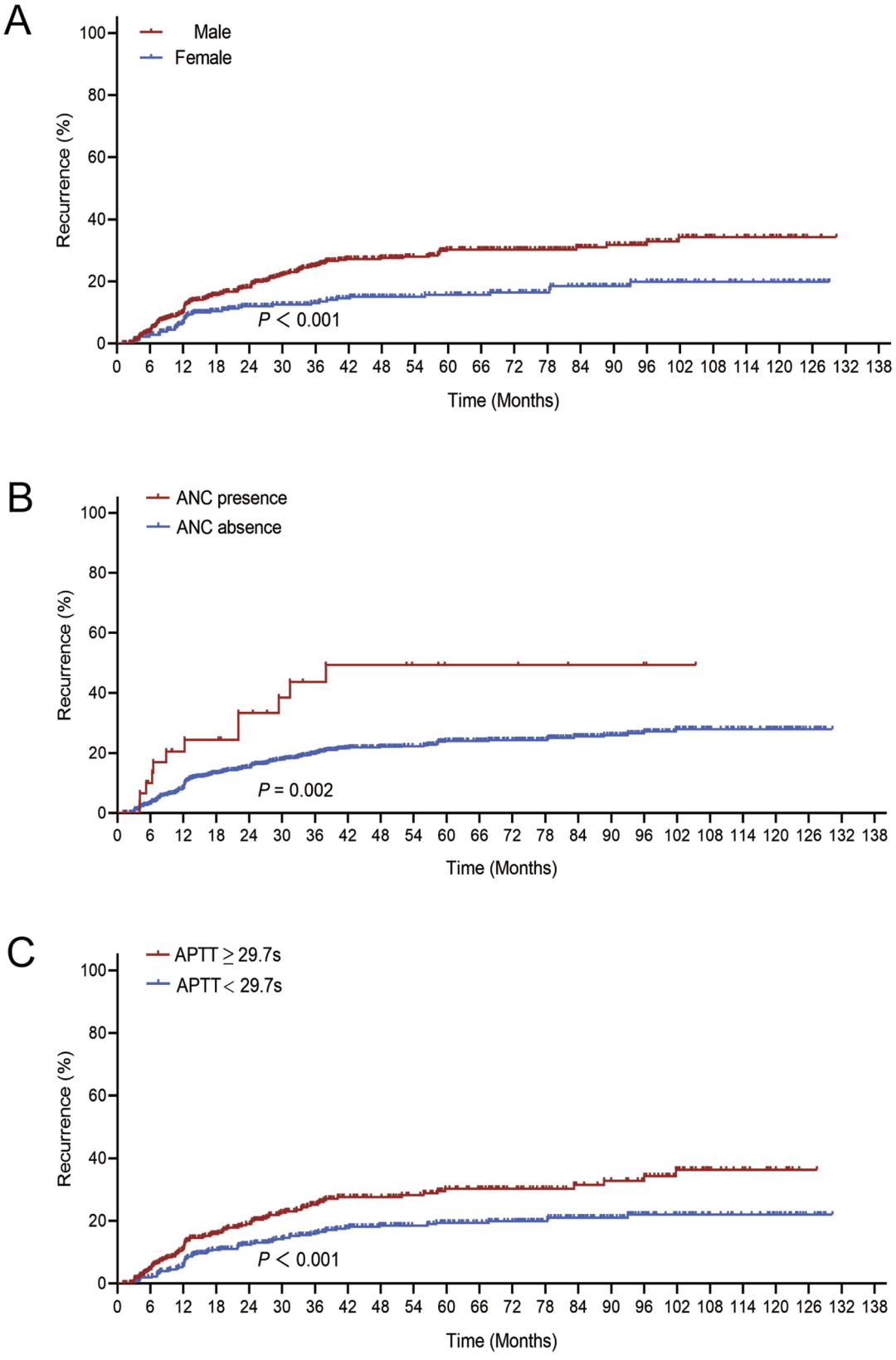

Fig. 3 Cumulative risk of the recurrence after the first episode of acute pancreatitis based on gender, ANC and APTT. (**A**) based on gender. (**B**) based on ANC. (**C**) based on APTT. ANC, acute necrotic collection; APTT, activated partial thromboplastin time

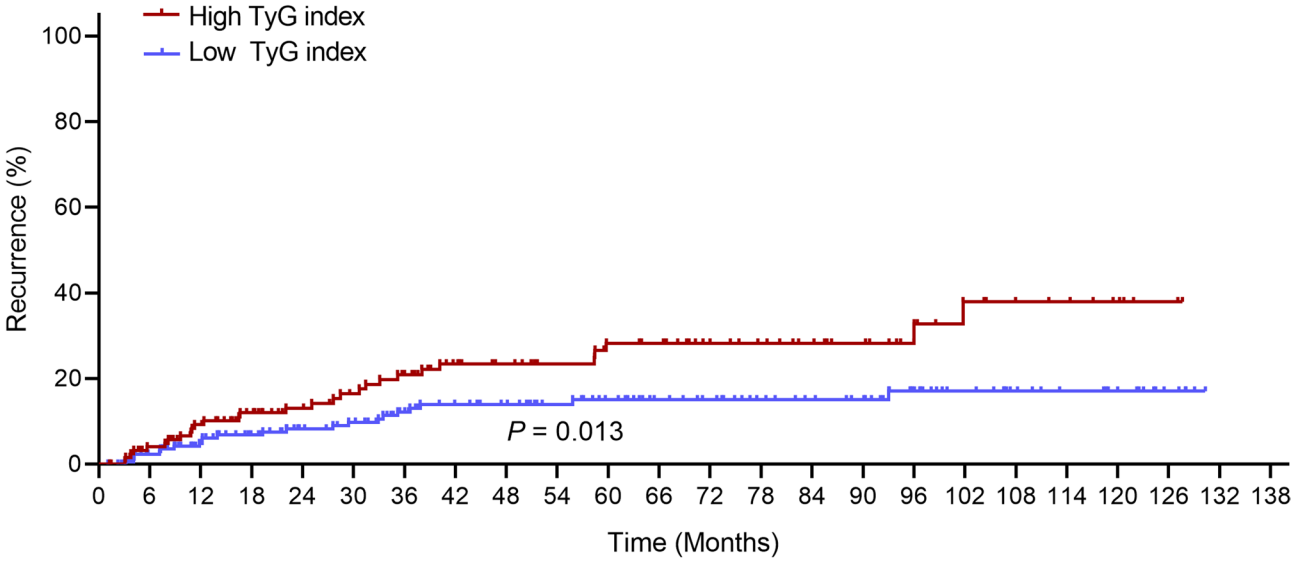

Fig. 4 Cumulative risk of the recurrence after the first episode of acute biliary pancreatitis based on the TyG-i. TyG-i, triglyceride-glucose index

TyG-i (HR = 1.996, 95% CI 1.146–3.476, $P = 0.015$) was a significant risk factor for the recurrence of acute biliary pancreatitis, and multivariate Cox regression analyses demonstrated that the TyG-i (HR = 1.829, 95% CI 1.043–3.025, $P = 0.035$) as an independent predictor of recurrent acute biliary pancreatitis (Supplementary Table 3).

General characteristics of RAP for all etiologies and biliary etiology

The general characteristics of RAP for all etiologies is shown in the Table 3. The median age of RAP in the high TyG-i group was 40 (34–49) years old, which was significantly younger than that in the low TyG-i group ($P = 0.019$). No statistically significant difference in local complication was observed between the two groups. The high TyG-i group had more systemic complications, including pulmonary/urinary infection ($n = 4$, 3.6%), intra-abdominal infection ($n = 2$, 1.8%), SIRS ($n = 2$, 1.8%), and shock ($n = 2$, 1.8%). Moreover, the high TyG-i group demonstrated higher WBC and neutrophil count, and higher levels of HB, NLR, APTT, and Fg (all $P < 0.050$; Table 3).

Similarly, the high TyG-i group at the recurrence of acute biliary pancreatitis demonstrated a significant increase in WBC count ($P = 0.005$), neutrophil count ($P = 0.008$), monocyte count ($P = 0.011$), and APTT time ($P = 0.004$), along with a significant decrease in platelet count ($P = 0.030$) (Supplementary Table 4). However, no statistically significant difference in local or systemic complication was observed between the two groups.

Discussion

Our study found that the TyG-i at the first episode of AP was a predictor of its recurrence. The TyG-i remained independently associated with the recurrence of acute biliary pancreatitis, which is the most common etiology. A significant correlation was observed between the TyG-i level and various laboratory indicators suggestive of inflammation when AP recurred. To the best of our knowledge, this is the first study to use the TyG-i, a simple marker, to evaluate the relationship between IR and AP recurrence.

IR is induced by inflammation and contributes to inflammation progression. Inflammation affects the roles of insulin in fat, skeletal muscle, and the liver. Serine kinases, such as c-Jun n-terminal kinase and inhibitor of kappa B kinase beta [19], well as cytokines, such as IL-1β, TNF-α, and IL-6 [20] directly led to IR. Moreover, metabolic pathways involving free fatty acids [21] and ceramide synthase [22, 23] affected insulin sensitivity. By contrast, specific knockdown of protein kinase B, a key kinase in the insulin signaling pathway, leads to IR and an increase in macrophages [24]. Mice with adipocyte–specific knockout of the serine threonine protein kinase PDK1, which is involved in insulin metabolism, developed IR and promoted liver inflammation [25]. After AP, IR increased, insulin clearance decreased [26]. The association between the first AP attack and IR development, as well as the mechanisms leading to disease recurrence, involves complex metabolic, immune, and endocrine processes. During the acute phase of AP, inflammatory factors, such as IL-6 led to islet cells damage and inflammatory responses, which may result in IR [27]. The IR-related mechanism that leading to recurrence after the first AP episode lacks extensive in-depth research.

Table 3 General characteristics of the recurrence of AP

Variables	Overall (*n* = 180)	Low TyG-i (*n* = 69)	High TyG-i (*n* = 111)	*P* value
Age (years)	42 (34–53)	46 (35–56)	40 (34–49)	0.019
Male (n, %)	134 (74.4)	46 (66.7)	88 (79.3)	0.059
WBC (×10^9/L)	10.9 ± 4.1	9.1 ± 3.3	12.1 ± 4.2	< 0.001
Neutrophil (×10^9/L)	8.3 (5.0-11.2)	6.2 (4.1–8.8)	9.6 (6.4–12.4)	< 0.001
Lymphocyte (×10^9/L)	1.5 (1.1-2.0)	1.6 (1.1-2.0)	1.5 (1.1–2.2)	0.649
Monocyte (×10^9/L)	0.5 (0.4–0.8)	0.5 (0.3–0.7)	0.6 (0.4–0.8)	0.087
HB (g/L)	146 (130–161)	136 (124–147)	155 (140–169)	< 0.001
RDWCV, %	13.0 (13.0–14.0)	13.0 (13.0–14.0)	13.0 (13.0–14.0)	0.971
PLT (×10^9/L)	229 (189–274)	218 (186–265)	230 (193–286)	0.547
NLR	5.8 (2.7–9.2)	3.8 (2.1–6.9)	7.0 (3.5–11.2)	0.002
APTT (s)	29.9 (27.4–33.5)	28.8 (25.9–32.6)	31.2 (28.0-35.4)	0.006
Fg (g/L)	3.5 (2.8–4.5)	3.0 (2.5–3.6)	3.9 (3.2–4.9)	< 0.001
Hospitalization length (Days)	5 (4–9)	5 (3–9)	5 (4–9)	0.355
ICU (n, %)	13 (7.2)	4 (5.8)	9 (8.1)	0.775
MCTSI				0.321
Mild (n, %)	99 (55.0)	40 (58.0)	59 (53.2)	
Moderate (n, %)	69 (38.3)	22 (31.9)	47 (42.3)	
Severe (n, %)	7 (3.9)	4 (5.8)	3 (2.7)	
Not assessable (n, %)	5 (2.8)	3 (4.3)	2 (1.8)	
Severity of AP				0.216
MAP (n, %)	144 (80.0)	59 (85.5)	85 (76.6)	
MSAP (n, %)	24 (13.3)	8 (11.6)	16 (14.4)	
SAP (n, %)	12 (6.7)	2 (2.9)	10 (9.0)	
Acute peripancreatic fluid collection (n, %)	26 (14.4)	9 (13.0)	17 (15.3)	0.673
Pancreatic pseudocyst (n, %)	18 (10.0)	6 (8.7)	12 (10.8)	0.646
ANC (n, %)	10 (5.6)	5 (7.2)	5 (4.5)	0.655
Walled-off necrosis (n, %)	13 (7.2)	4 (5.8)	9 (8.1)	0.775
Infected pancreatic necrosis (n, %)	2 (1.1)	1 (1.4)	1 (0.9)	1.000
Ascites/peritonitis (n, %)	32 (17.8)	13 (18.8)	19 (17.1)	0.769
Portal hypertension (n, %)	12 (6.7)	4 (5.8)	8 (7.2)	0.951
Other systemic complications (n, %)	24 (13.3)	5 (7.2)	19 (17.1)	0.058

Note: Values are presented as means ± standard deviation, median (interquartile range) or n (%). Mild (MCTSI score 0 and 2), moderate (MCTSI score 4 and 6), severe (MCTSI score 8 and 10). Other systemic complications refer to intra-abdominal infection, stroke, coronary heart disease, ARDS, AKI, SIRS, pulmonary/urinary infection, gastrointestinal bleeding, MODS, septicemia, abdominal compartment syndrome, pancreatic encephalopathy, intestinal obstruction, shock

Abbreviations: AP, acute pancreatitis; TyG-i, triglyceride-glucose index; WBC, white blood cell; HB, hemoglobin; RDWCV, red blood cell distribution width coefficient of variability; PLT, platelet; NLR, the ratio of neutrophil to lymphocyte; APTT, activated partial thromboplastin time; Fg, fibrinogen; ICU; intensive care unit; MCTSI; modified CT severity index; MAP, mild acute pancreatitis; MSAP, moderately severe acute pancreatitis; SAP, severe acute pancreatitis; ANC, acute necrotic collection

Currently, many methods are available to evaluate IR, each with different validity and reliability. The hyperinsulinaemic euglycaemic clamp technique, which is recognized as the gold standard for detecting IR, requires continuous insulin infusion [28]. Detection of fasting plasma insulin can directly reflect IR; however, individuals with impaired islet function may require further detection of postprandial plasma insulin or response under the load of glucose [29, 30]. The homeostasis assessment model, calculating by fasting blood glucose and fasting blood insulin, is not suitable for patients with severe hyperglycemia or pancreatic β cell failure [29, 30]. The clinical management of AP seldom pay attention to plasma insulin level. TyG-i combines fasting blood glucose and triglyceride levels during routine examinations. It is a simple, convenient, and low-cost method for detecting IR, and is appropriated for various subjects [31]. Therefore, it is feasible and reliable to measure the levels of fasting blood glucose and triglyceride during the first-episode of AP and calculate the TyG-i level to evaluate the IR status.

A cross-sectional study showed that most RAP cases occurred in men [32]. Other studies have indicated that male sex as an independent risk factor for RAP [33, 34], which is consistent with our findings. A retrospective study has reported that ANC could predict AP recurrence [2]. Similarly, ANC was significantly associated with RAP in our cohort. A prolonged APTT often indicates an endogenous coagulation disorder and increased bleeding risk. Several studies have reported that APTT

is a risk factor for AP-related organ failure and mortality [35, 36, 37]. The Cox proportional hazards analysis and survival analysis we performed showed that a long APTT could predict AP recurrence. Pro-inflammatory cytokines in AP activated the vascular endothelium, triggered the coagulation cascade, reduced anticoagulation, inhibited fibrinolysis, and stimulated microvascular thrombosis. Coagulation and fibrinolysis markers were correlated with the severity and prognosis of AP [38, 39, 40].

In our cohort, RAP with higher TyG-i level was associated with a higher incidence of complications and abnormal laboratory examination results, whereas no statistically significant difference was observed in the cases of severe MCTSI and SAP. As most patients with mild RAP, TyG-i had a low ability to distinguish severity. Severe RAP cases accounted for 10.1% of the cases in our study compared to 10.7% [33], 18.5% [41], and 12% [42] in other studies.

In our study, the stroke incidence in the low TyG-i group was higher than that in the high TyG-i group. Although this difference was not statistically significant, it was sufficient to attract our attention. TyG-i, as a marker of IR and systemic inflammation, is a predictor of cardiovascular diseases. The middle-aged and elderly Chinese population with higher TyG-i levels had a higher overall cardiovascular disease risk, including stroke [43]. In patients with acute coronary syndrome and high TyG-i levels, the occurrence of adverse cardiovascular events, including stroke, was increased after percutaneous coronary intervention [44]. In our study, the patients in the low TyG-i group were significantly older than those in the high TyG-i group, and the proportion of female patients was significantly higher. A hospital-based prospective observational study has reported that the number of female patients with stroke was significantly higher than that of male patients [45]. Thus, the higher stroke incidence in the low TyG-i group may have been influenced by common stroke risk factors, such as age and gender. LMWH is an anticoagulant used to treat various clinical conditions. In addition, LMWH has potent anti-inflammatory property [46, 47], which are believed to play a key role in improving the prognosis of AP. Moreover, LMWH can rapidly reduce blood triglyceride levels [48, 49]. All the effects of LMWH (anticoagulation, anti-inflammatory, and lipid-lowering) may help suppress the adverse outcomes of AP. Based on the evidence-based medicine, our center uses LMWH treatment in patients to provide anti-inflammatory effects or rapidly lower blood triglyceride levels.

Our study has some limitations. First, this was a retrospective study that lacked useful and emerging pieces of information, such as waist circumference (WC) and waist-hip ratio (WHR), which can be combined with TyG-i as TyG-WC and TyG-WHR. Multicenter prospective studies are required in the future to confirm and improve our findings. Second, only blood glucose and blood triglycerides within 24 h after admission were collected, and data during the course of disease were not included. It is expected to provide a new direction to study on the relationship between the dynamic changes in TyG-i and the severity, efficacy, and prognosis of AP. Nevertheless, our results could serve as a reference for future clinical studies and clinical management.

Conclusion

The TyG-i at the first AP episode is an independent predictor of the recurrence.

Abbreviations

AP	Acute pancreatitis
RAP	Recurrent acute pancreatitis
IR	Insulin resistance
TNF-α	Tumor necrosis factor-α
IL-6	Interleukin-6
TyG-i	Triglyceride-glucose index
CT	Computed tomography
ICU	Intensive care unit
MCTSI	Modified computed tomography severity index
MAP	Mild acute pancreatitis
MSAP	Moderate severe acute pancreatitis
SAP	Severe acute pancreatitis
SIRS	Systemic inflammatory response syndrome
APTT	Activated partial thrombin time
Fg	Fibrinogen
LMWH	Low-molecular-weight heparin
CRRT	Continuous renal replacement therapy
WBC	White blood cell
HB	Hemoglobin
NLR	The ratio of neutrophil to lymphocyte
HR	Hazard ratio
CI	Confidence interval
HR	Hazard ratio
CI	Confidence interval
ANC	Acute necrotic collection
WC	Waist circumference
WHR	Waist-hip ratio

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12986-025-00956-7.

Supplementary Material 1

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Not applicable.

Author contributions

Lihui Lin designed the project and conducted data collection and analysis. Yansong Lin contributed to article revision. Xin Ling and Zewen Zhang provided valuable advices. Xianwen Guo and Ding Zhen provided financial support and supervised this work. All authors read and approved the final manuscript.

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Data availability
The datasets that support the findings of this study are available from the corresponding authors on reasonable request.

Declarations

Ethics approval and consent to participate
This study was performed in line with the principles of the Declaration of Helsinki and was approved by the Clinical Research and Laboratory Animal Ethics Committee of the First Affiliated Hospital of Sun Yat-sen University (Date July 4, 2024/No. [2024]495).

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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